REMARKS

Claims 10-15 are active in the present application. Support for the amendment to Claim 10 is found in Claims 6 and 7, and the specification as originally filed. No new matter is added. Favorable reconsideration is respectfully requested.

Applicants wish to thank Examiner McGarry for the courteous discussion held with the undersigned Applicants' representative on October 19, 2000. The subject of this discussion is summarized in the remarks made below.

The rejection of Claims 1-8 under 35 U.S.C. §102(a) over Russell et al is obviated by the cancellation of these claims.

The rejection of Claim 9 under 35 U.S.C. §103(a) over Russell et al in view of Stull et al is obviated by the cancellation of this claim.

The rejection of Claims 6-9 under 35 U.S.C. §112, second paragraph, is obviated by the cancellation of these claims.

The rejection of Claims 1-6 and 8-16 under 35 U.S.C. §112, first paragraph, is respectfully traversed.

The instant claims recite a method for treatment of NF-κB associated disease which comprises administering to an animal a polynucleotide comprising the 8th through the 17th nucleotide of SEQ ID NO:1. The specification provides adequate support and enablement for the use of such a polynucleotide by antagonizing NF-κB-mediated transcription region located downstream of a NF-κB binding site. The instant specification provides examples of using this polynucleotide to treat myocardial infartions, cancer metastasis, and inhibition of cachexia (see Examples 1-5, pages 12-18). In addition, Applicants provide herewith a number of published or articles currently in press, authored by the present inventors, which demonstrate the success of using NF-κB decoys (e.g., those containing the 8th through the

17th nucleotides of SEQ ID NO:1) for treating a variety of NF-κB associated diseases (see attached PTO Form-1449). For example, the attached references demonstrate successful treatment of myocardial cell infiltration , arterial neointimal formation, ischemia, synovial cell inhibition, restinosis after angioplasty, cerebral angiopathy, TNF inhibition, inflammation, atherogenesis, arthritis, glomerular inflammatory response, cresentic glomerulonephritis, intimal hyperplasia, and autoimmune myocarditis. These and other diseases are described in the specification as those which are associated with NF-κB (see page 3). Thus, in view of the data presented in the specification and the subsequent publications which, based on the teachings of the present specification, demonstrate the effectiveness in treating NF-κB associated diseases fully enables the scope of the claimed method. Accordingly, withdrawal of this ground of rejection is respectfully requested.

Applicants submit that the present application is now in a condition for allowance.

Early notification of such is earnestly solicited.

Respectfully submitted,

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